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# SELENIUM ROLE IN AN HUMAN BIOMONITORING STUDY APPLIED TO OCCUPATIONAL HEALTH



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## Introduction

Selenium functions as a co-factor for the reduction of antioxidant enzymes and is an important component of antioxidant enzymes. Dietary selenium significantly inhibits the induction of skin, liver, colon, and mammary tumours in experimental animals by a number of different carcinogens, as well as the induction of mammary tumours by viruses. Selenium shows a “U” shaped curve for functionality, whereby too little is as damaging as too much. At optimal levels, selenium may protect against the formation of DNA adducts, DNA or chromosome breakage, chromosome gain or loss, mitochondrial DNA, and telomere length and function.

## Aim of the Study

Investigate the relation between selenium and genotoxic effects in a human biomonitoring study applied to occupational health.

## Results

Selenium item was measured by Food Frequency Questionnaire (FFQ). The quantification of the dietary parameters for this is shown in Table 1.

**Table 1** –Dietary parameters of selenium measured by FFQ (mean intake per day ± standard deviation) and respective dietary reference intakes.

Parameters	Groups	Mean± Std. Deviation (daily nutrient intake)	Dietary References Intakes (Food and Nutrition Board, Institute of Medicine, National Academies) d= day
Selenium	Exposed	131.51±9.34	45 µg/d
	Controls	138.67±8.58	

## Methodology

The study was carried out in Portugal in a sample of 46 subjects exposed occupationally to cytostatics and 46 subjects without any occupational exposition. Blood samples were taken and used to measure genetic instability biomarkers represented by DNA damage breaks and oxidative stress. The evaluation of genotoxic effects was conducted by applying two techniques: cytokinesis-blocked micronucleus assay (CBMN) and comet assay in peripheral blood lymphocytes. CBMN allowed to measure DNA damage by quantifying micronucleus (MN), nucleoplasmic bridges (NPB) and nuclear Buds (NBUD). Comet assay measured DNA breaks (% DNA in tail) and oxidative DNA damage (FPG). A food-frequency questionnaire was fulfilled, and the selenium intake calculated using the FREQUAN dietary analysis program.

**Table 2** - Spearman correlations between MN, NPB, NBUD, % DNA in tail and oxidative DNA damage (FPG) and selenium item in the exposed (A) and the control (B) groups. No significant correlations were signaled.

**A – Exposed**

	Selenium
MN lymph	-0.053
NPB	-0.037
NBUD	-0.148
% DNA TAIL	0.073
FPG	-0.069

**B – Non-Exposed**

	Selenium
MN BN	-0.096
NPB	-0.143
NBUD	-0.063
% DNA TAIL	-0.222
FPG	0.074

It was not found any statistical significant association between the genotoxicity biomarkers studied and selenium in each group. For both groups, the increase of selenium intake was related with the decrease of genomic instability, however without reaching statistical significance. Current dietary recommendations do not consider the concept of genome stability which is of concern because damage to the genome has been linked to the origin and progression of many diseases and is the most fundamental pathology.